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Special Review

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Post-Traumatic Craniocervical Disorders From a Postural Control Perspective: A Narrative Review

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HIGHLIGHTS

- Post-concussive syndrome (PCS) or whiplash-associated disorders (WAD) can affect head and neck postural sensors, such as the ocular sensorimotor, vestibular, and cervical proprioceptive systems.
- Symptoms observed in PCS or WAD are also found in cases of non-traumatic impairment of postural sensors, indicating the need for careful attention to these structures.
- Several therapeutic interventions targeting traumatic postural sensors impairment have been attempted, with some showing promising results

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Post-Traumatic Craniocervical Disorders From a Postural Control Perspective: A Narrative Review

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ABSTRACT

Mild traumatic brain injury (mTBI) and whiplash injury (WI) may lead to long-term disabling consequences known as post-concussive syndrome (PCS) and whiplash-associated disorders (WADs). PCS and WAD patients commonly complain of conditions encompassing dizziness, vertigo, headache, neck pain, visual complaints, anxiety, and neurocognitive dysfunctions. A proper medical work-up is a priority in order to rule out any acute treatable consequences. However investigations may remain poorly conclusive. Gathered in the head and neck structures, the ocular sensorimotor, the vestibular, and the cervical proprioceptive systems, all involved in postural control, may be damaged by mTBI or WI. Their dysfunctions are associated with a wide range of functional disorders including symptoms reported by PCS and WAD patients. In addition, the stomatognathic system needs to be specifically assessed particularly when associated to WI. Evidence for considering the post-traumatic impairment of these systems in PCS and WAD-related symptoms is still lacking but seems promising. Furthermore, few studies have considered the assessment and/or treatment of these widely interconnected systems from a comprehensive perspective. We argue that further research focusing on consequences of mTBI and WI on the systems involved in the postural control are necessary in order to bring new perspective of treatment.

Keywords: Whiplash Injury; Chronic Post Concussive Syndrome; Postural Control; Dizziness; Stomatognathic Disease

INTRODUCTION

Patients that underwent whiplash injury (WI) and mild traumatic brain injuries (mTBIs) exhibit a wide range of complaints with small discrepancies between the 2 entities [1]. The resulting conditions are known as post-concussive syndrome (PCS) and whiplash-associated disorder (WAD). They include subjective complaints such as dizziness, visual disorders, cognitive impairment, headaches and neck complaints [1-3]. PCS is a prevalent condition

following mTBI, the most common type of TBI [4,5]. Literature shows that 30% to 80% of patients will exhibit symptoms in the following days and up to 20% after 10 years post trauma [2,6]. Similar observations have been reported in WAD patients [7-9].

In both types of injuries, kinetic energy is shifted into mechanical energy which is subsequently dissipated in the craniocervical structures [10]. Resulting lesions encompass diffuse axonal injury, cerebral contusions, subdural hematoma and subarachnoid hemorrhages, fractures, or cervical spine sprain easily detectable by classical radiological work up [11-13]. Less evident alterations such as metabolic, physiologic and microstructure changes undetectable by usual medical investigations may also result from the craniocervical trauma (CCT) [6,14]. Functional imagery, currently used in research, may provide new comprehensive tools. This is especially true for cognitive impairments following CCT [15-17] but remains poorly investigated for other symptoms. Most symptoms reported by PCS or WAD patients still lack satisfactory support from investigations. Recommendations on how to treat the resulting conditions suffer from a substantial lack of evidence [18]. Moreover, pre-existing anxiety and depression and post-traumatic stress disorder may affect the expression of symptoms and account for a cause of prolonged PCS and WAD recovery [19-22]. Including diagnosis, proper treatment and follow-up of such conditions are therefore needed in the management of patients presenting long term consequences of mTBI and WI [23].

Noteworthy, impaired postural control is commonly observed after CCT, as suggested by the high prevalence of following balance disorders and vestibular-like symptoms such as vertigo and dizziness [24-27]. Gathered in the head and neck, the vestibular and the ocular sensorimotor systems as well as the cervical spine proprioceptors, may be damaged during the trauma. Concussion rating scales poorly assess the consequences of CCT on these structures [1,28,29] with visual, oculomotor and vestibular system impairment being the most represented aspects of such deficiencies [28]. While the topic remains controversial, growing evidence supports the role of the stomatognathic system in postural control [30-33]. It is reportedly also a potential victim of CCT, especially when WI is associated [34,35].

A broad range of functional symptoms similar to those noticed in PCS or WAD may result from impairment of vestibular, visual, cervical proprioceptive and stomatognathic systems. This article proposes a comprehensive review targeted on post-CCT dysfunction of these systems and their management from a postural control perspective.

SEARCH STRATEGY

A comprehensive review of published literature on PubMed, Scopus and Google Scholar was achieved up to December 2022. We focused our search on the consequences of CCT on cervical spine, vestibular system, visual and oculomotor systems and the stomatognathic system. We implement corresponding MeSH terms of these systems with “post concussive syndrome,” “mild traumatic brain injury,” whiplash injury” or “whiplash associated disorder.” Preferential consideration to systematic reviews and randomized control trials was given when possible. The literature probed in each research subsection was assessed by 3 main types of symptoms: headache, neck pain and dizziness/vertigo.

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POST TRAUMATIC CRANIOCERVICAL DISORDERS REVIEWED FROM A POSTURAL CONTROL PERSPECTIVE

Growing literature is currently focused on the evaluation and treatment of the impairment of the craniocervical structures involved in postural control after CCT. New treatment perspectives may result from such consideration in PCS and WAD. Evidence are developed in this section.

Post-traumatic ocular sensorimotor system impairment

The ocular sensorimotor system includes vision and the oculomotor system. Visual complaints after CCT, even in the absence of direct ocular trauma, have been reported for over a century [2,24]. Ocular sensorimotor dysfunction has also been associated with symptoms such as headaches [36], neck complaints [24] and dizziness [37].

Post-traumatic ocular sensorimotor impairment and associated visual complaints

Visual symptoms may arise from primary vision deficit, eye movement disorders, and more complex vision-associated functions. Symptoms such as diplopia, visual acuity loss, blurred vision, photophobia, loss of color vision, stereopsis deficit, impaired visual accommodation and visual field impairment have been reported after CCT [38-40]. The main functions of the ocular sensorimotor system are described in **Fig. 1** [41]. Sensory and motor ocular functions provide an accurate depiction of a visual target as well as a more global spatial representation of the surrounding environment. Complex connections exist between this system, the vestibular system and the axial musculoskeletal structures. They allow gaze orientation and stabilization as well as head and trunk stabilization in accordance with visual inputs.

TBI, including mTBI, may result in a wide range of ocular sensorimotor dysfunctions reported in **Table 1** [38,39,42-46]. Convergence insufficiency seems to be the most frequent oculomotor deficit following mTBI affecting up to 42% of patients [47,48]. It may be an

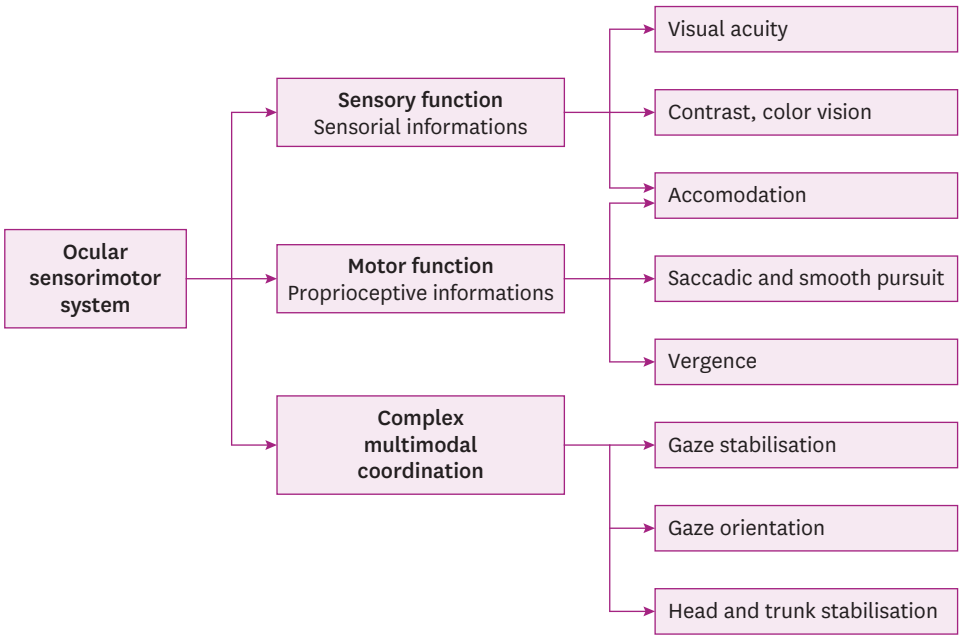

Fig. 1. Inspired from Pipet and bertagnolio [41] ocular sensorimotor system functions. All of these functions may be impaired following craniocervical trauma and potentially responsible for visual complaints.

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Table 1. Reported ocular sensorimotor dysfunctions after craniocervical trauma

Dysfunctions
- Visual acuity and visual fields impairments [38]
- Eye movement disorders: accommodation/vergence disorders, saccadic pursuit, smooth pursuit and cranial nerve palsy (III, IV, VI), nystagmus [38,39,42-44]
- Accommodation and pupillary dysfunctions and pseudo myopia [38,45]
- Visual perception, motion vision, and visuo-spatial functions [38]
- Gaze stabilisation and dynamic acuity [46]

early clinical sign following TBI [38]. This major function of the visual system is needed to focus the eyes on a target at varying distances. It is closely associated to the function of eye accommodation. Convergence insufficiency may result from direct cortical, subcortical, or brainstem visual/ocular network impairment [49]. Furthermore, some authors suggest that oculomotor deficits may be considered as biomarkers of brain network dysfunction as they are more prevalent in patients with higher cognitive loads after mTBI [50].

Multiple ocular sensorimotor dysfunctions may also follow WI or other neck trauma. The relationship between neck pain, a frequent condition following WI [51], and subjective visual complaints are nowadays well investigated [24,52-54]. Evidence suggests that cervical proprioceptive system impairment may result in oculomotor dysfunctions [24]. Impairment in smooth pursuit, gaze stability, convergence, saccade, and head coordination have indeed been reported after a WI [24]. However particular attention must be given to vestibular structures as their dysfunction may also lead to oculomotor disorders [24]. Dysfunctions of the vestibular and visual structures may conversely impair the cervical spine's range of motion and induce musculoskeletal neck pain [24,55].

Promising interventions have been proposed for visual disturbances following mTBI. However consensus and evidence regarding these treatments is still lacking [56-59].

More precisely, specific interventions to treat visual acuity, color vision impairment and photophobia associated with mTBI have demonstrated a potential efficacy [38]. Other treatments such as vertical prisms, filters, nasal occlusion and vestibular exercises have also been attempted with however less supported benefits [59]. Also, while convergence insufficiency or accommodative rehabilitation can help with visual complaints in non-traumatic patients, their rehabilitation after mTBI remains poorly documented [58] but promising [60]. Some authors such as Treleaven [24] suggest a combined approach including oculomotor consideration in association with exercises targeting deficits in cervical joint position, and static and dynamic imbalance.

As visual deficits may result in headaches, poorer concentration, fatigue, daily life activity difficulties, and overall reduced quality of life [57], clinicians should consider including a visual and oculomotor examination in post-CCT patients.

Post-traumatic ocular sensorimotor impairment and other associated conditions

Ocular sensorimotor disturbances following CCT are often associated with non-specific conditions such as dizziness and headaches.

Vestibular symptoms such as dizziness and feeling of imbalance are common conditions following CCT [37]. There seems to exist a direct correlation between the severity of convergence insufficiency, concussion and vestibular system impairment [48]. Proposed

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explanations rely on the close interconnections existing between these systems [48]. D'Silva and colleagues [61] also found in 22 post-mTBI patients a significant association between oculomotor deficits and poorer dynamic mobility and dizziness. Oculomotor deficits have also been linked with prolonged recovery after sport-related concussions [62, 63]. Treatment of convergence insufficiency in non-post-traumatic subjects has been associated with improved postural control in a small interventional study [64] and restored normal postural behavior in another study [65]. However combining both visual-oculomotor and vestibular therapies to alleviate dizziness in post-concussion patients still lacks evidence [37].

Post-traumatic headache is the most common sequelae following brain injury and may last for years after the trauma [66]. This condition may also result, in a substantial part, from WI [51,67] and may present itself as tension-type headaches and migraine-like symptoms [68]. Ocular causes of headaches include 1) refractive errors, 2) convergence insufficiency, and 3) accommodative spasm [36]. Ocular headaches are mainly frontally localized and more severe at the end of the day. They are also more frequent in patients performing near work (i.e., work on screen) [36]. However, although non traumatic convergence insufficiency and accommodative rehabilitation therapy seems to bring promising benefits [38,58,59], this role needs to be further evaluated in CCT patients.

Post-traumatic vestibular system impairment

The vestibular system, which includes the semicircular canals, the otolith system, the vestibular nerve, the vestibular nuclei and the related subcortical and cortical networks, may be damaged by CCT. Subsequently vestibular disturbances commonly result in vertigo, dizziness, or feeling of imbalance. These conditions are commonly observed after CCT [2,25,69,70].

Post-traumatic vestibular associated diseases

Benign paroxysmal positional vertigo, labyrinthine concussion, and vestibular migraine are the most common etiologies of peripheral vestibular system disorders reported after mTBI [25]. Direct vestibular nerve or labyrinth injury resulting from petrous bone fracture, potentially generating a perilymphatic fistula, has also been associated with CCT to a lesser extent [70,71]. More recently, a case series pointed out that CCT may also potentiate superior semicircular canal dehiscence syndrome [72]. **Table 2** sums up the vestibular disorders reported after CCT [25,70-72].

Vestibular impairments however seem to be more frequent after WI than TBI [1]. They remain underestimated, often being incorrectly attributed to cervical or cerebral lesions [73]. Central vestibular impairment is mostly secondary to injuries to the vestibular nuclei or central vestibular pathways. As reported for ocular motor dysfunction, mTBI may result in brainstem concussion and potentially damage the vestibular nuclei. Cortical and subcortical vestibular pathways may also be injured by the traumatic mechanisms [70,74].

Table 2. Reported vestibular disturbances after craniocervical trauma

Vestibular disturbances
- Benign paroxysmal positional vertigo [25,70]
- Labyrinthine and vestibular nerve injury [25,70,71]
- Vestibular migraine [25,70]
- Perilymphatic Fistula [70,71]
- Ménière disease or endolymphatic hydrops [71]
- Superior semicircular canal dehiscence syndrome [71,72]

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The vestibular system is one of the main pillars of postural regulation [75]. It is intimately linked to the visual system through the vestibulo ocular reflex. The vestibulo ocular reflex is essential to gaze stabilization, dynamic visual acuity, and the development of visuospatial perception during head motion. It is estimated to be altered in approximately 50% of mTBI patients [46]. This association is however less documented for WAD [52].

Vestibulospinal bundles emerge from vestibular nuclei and enable the association between the vestibular system and body posture. Complex connections exist between these structures, the cerebellum and the reticular formation involved in postural control [46,76]. They are responsible for maintaining the body's orientation in space and contribute to postural tone. The vestibulospinal reflex represents the bridge between head and cervical spine coordination since it participates in stabilizing the head on the neck [46].

Post-traumatic vestibular impairment and related symptoms

About half of post-TBI patients experience dizziness that may last up to 5 years following mTBI [25]. Same observations have been made in a substantial amount of post-WI patients [69]. Dizziness following concussion is an important symptom to assess as it stands as a predictor of prolonged recovery [77]. However, feelings of imbalance and dizziness may reveal a predominant psychogenic component when they persist [71]. This symptom may also result from cervical proprioception impairment [24]. It has also been significantly associated with oculomotor dysfunction following CCT [61]. It is notably estimated that dizziness remains unassociated with a vestibular or a detectable central dysfunction in approximately a quarter of PCS patients [78].

Vestibular rehabilitation therapy (VRT) seems to constitute a promising approach to treat dizziness and imbalance following CCT [79]. Stronger evidence are still needed [80, 81].

Studies that examined the efficacy of VRT in PCS patients with dizziness documented significant improvement in self-perceived dizziness [27,77] and balance [27]. Concerning dizziness following WI, a small cross-sectional study reported a significant improvement in dizziness among the 20 patients that received VRT [69]. A randomized control trial run on 29 patients presenting WAD also reported a decrease in self-perceived handicap and an increase in postural control among patients performing VRT [81]. Finally, initiating VRT early in sport-related concussions has been associated with an earlier return to activity and symptom resolution [82].

Visual dependency is a condition in which patients rely preferentially on visual inputs to ensure balance. This condition may be responsible for inappropriate gait and balance strategies following TBI [83, 84].

We assumed that visual dependency could also result from self-perceived imbalance disorder after CCT. On the one hand, PCS patients seem to rely more on visual and vestibular inputs to control balance than do patients without PCS [85]. On the other hand, these patients may have been exposed to vestibular peripheral system dysfunction triggering visual dependency. For these reasons, we suggest that visual dependency remains an underestimated condition following CCT.

Post-traumatic cervical spine impairment

Cervical fracture, sprain, or muscle injury are common in CCT. While investigations of the cervical spine may remain non-contributive, complaints concerning neck pain are extremely

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frequent after CCT [24,86,87]. Other symptoms such as dizziness, vertigo, and headaches have also been reported as potential consequences of cervical spine impairment.

Post-traumatic cervical pain

Neck pain is a highly prevalent, yet underdiagnosed, disabling condition after WI [87] and in acute and chronic stages of PCS [88]. According to a recent meta-analysis, 84% of post-WI patients experience neck pain in the week and up to 38% in the year following the trauma [60]. Neck pain after mTBI seems to be more common after motor vehicle collisions than in other mechanisms of injury [87].

Usual medical investigations for neck pain after CCT remain poorly contributive or examined. MRI investigation, one of the most sensitive imaging techniques to detect signs of muscular related alterations, remains inconclusive in most WAD [89]. Some authors also evaluated the usefulness of ultrasound studies with promising results. When compared to the control group, Rahnama and colleagues [90] found a significant prevalence of neck muscular deformation associated with neck pain in 36 WAD patients. Kalawy and colleagues [91] using color doppler, reported an increased number of high blood flow in painful regions in 20 WAD patients with neck pain compared to the control group. However no further decisive studies were carried out despite these 2 findings. Therefore neck pain remains mostly subjective in WAD patients.

Numerous interventions regarding post-traumatic neck pain have been attempted so far. Manual therapy including mobilization, manipulation, and clinical massage for treating neck pain remain the most examined and useful interventions [92]. However, clinicians should always consider a multimodal approach including patient education, range of motion reassurance, strengthening exercises, and multimodal care in a complete consideration of post-CCT neck pain [24,93].

Post-traumatic cervical and associated disorder

As previously discussed dizziness and vertigo are common after CCT but remain cryptogenic in 25% of reported cases [25]. A quite recent, but still discussed entity, namely “cervicogenic dizziness” (CD) [94] also known as “cervicogenic vertigo” [9] supports the currently underestimated role of the cervical spine in postural control. Although CD patients commonly exhibit impairment in neck range of motion as well as neck pain [94,95], CD remains a diagnosis of exclusion [95].

The cervical spine plays role in determining the position of the head on the trunk along with the visual and the vestibular systems [94,96]. Cervical zygapophyseal joints, especially from C1 to C3, and cervical muscles are widely provided with proprioceptors and mechanoreceptors, anatomically supporting that role [97]. Evidence from animal studies demonstrated the existence of close connections between vestibular nuclei and cervical proprioceptors potentially explaining eye, head, and neck coordination, perception of balance, and postural adjustments [9]. Dysfunction in sensorimotor information arising from cervical spine may result from traumatic, degenerative or other forms of cervical injury. The mismatch between the visual system and misleading cervical sensorimotor information is assumed to result in abnormal central postural integration and therefore in vertigo or dizziness [9,97].

There is currently no strong evidence for which therapy might be the most useful to manage CD especially after CCT. Manual therapy has been investigated in non-traumatic related

CD and seems promising [98,99] even more when combined with exercise therapy [99]. Retraining deep cervical flexor and extensor muscles also participates in reducing neck pain and improving cervical proprioception [96]. Therapies that reduce neck pain may also improve postural symptoms. This reduction is thought to occur though the decrease of sensitivity of cervical spine nociceptors and proprioceptors [96]. Some authors proposed a multimodal approach based on the close connections linking vestibular, ocular motor, and cervical sensorimotor systems. We assume that in neck pain, whether post-traumatic or not, neck dysfunction must be considered as a potential cause, as well as any cause that results in impaired postural control.

Cervicogenic headaches are an increasingly recognized entity [100]. This entity may be tricky to clinicians as it shows sometimes migraine or classical tension headache features [101]. Post-traumatic headaches seem to benefit more from a multimodal approach combining pharmacological and non-pharmacological interventions, which encompass noninvasive neuromodulation, physical therapy, cognitive-behavioral treatment, and education [102], yet, evidence remain weak on the topic [103].

Post-traumatic stomatognathic impairment

The stomatognathic system encompasses teeth, mandible and other associated soft tissues. The hyoid bone and the tongue are also included in the system. We present in this section evidence for considering stomatognathic disorders in the evaluation of post-traumatic disorders.

Trauma related stomatognathic lesions

The stomatognathic system is not usually assessed after CCT. Lesions of its components can result from any kind of head and neck trauma [104-109], even if they are more frequently associated with WI [34, 108-110].

Patients commonly report symptoms and exhibit clinical signs suggestive of temporomandibular disorder (TMD) after WI. These reported symptoms suggest the necessity of assessing not only the temporomandibular joint but also other cervical joints subsequent to such trauma [51].

At follow-up, Salé and Isberg [111] reported an incidence of TMD pain in approximately one-third of post-whiplash patients compared to 7% in the control group at one year. They also showed a significantly increased frequency of temporomandibular joint pain in females than in male subjects [111]. Häggman-Henrikson et al. [34] in a systematic review based on 8 studies, reported a range of incidence of TMD pain between 4% to 34% after WI compared to 4.7% to 7% in the control group.

Many post-traumatic lesions of the temporomandibular joint and its related soft tissue may contribute to TMD after WI. For instance, Lee and colleagues [108,109] reported in 2 case-control studies an increased prevalence of disk deformity and atrophy of the lateral pterygoid muscle after whiplash compared to control.

The stomatognathic system and postural control

Growing evidence suggests a relationship between the stomatognathic system and the postural control [31-33]. This role remains, however, poorly understood, and controversial [112]. The anatomical connections between trigeminal sensory and vestibular nuclei could support this relationship [31]. Others remind the interconnections between trigeminal

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sensory system and reticular formation suggesting that excessive proprioceptive inputs arising from the stomatognathic system may induce interferences with efferent motor cortex copy of movement, leading to imbalance disorders [33]. The contribution of the stomatognathic system seems however more important when facing difficult conditions such as unstable support surface, fatigue or during specific task realization [31,113].

Stomatognathic system dysfunction has been linked with the impairment of the other head and neck systems involved in the postural control.

In addition to their shared sensorial afferent information on the sensory trigeminal nucleus, evidence supports close interconnections between the stomatognathic and the ocular sensorimotor systems [114]. Sharifi Milani and colleagues [30] first reported variations in the Maddox rod test, looking for vertical heterophoria, and Berens prismatic bars depending on whether the patient wore a mandibular orthopedic repositioning appliance. Monaco and colleagues [115] observed that ocular convergence insufficiency was more likely to affect patients with mandibular lateral deviation than controls. Cuccia and Caradonna [32] found similar observations in patients with temporomandibular joint displacements. Vompi and colleagues [116] also reported on 100 TMD patients a significant increase in ocular convergence insufficiency but also ocular deviation such as phorias and tropias as well as astigmatism compared to controls. Similar observations were made with dental malocclusion concerning tropia, phoria, ocular convergence defects, astigmatism, and myopia [117]. It seems, therefore, undeniable that direct neurophysiological connections exist between both systems. However, the exact pathways that supports this relation remain to be investigated.

Vestibular-related symptoms such as vertigo and dizziness are frequent in TMD patients [118]. However, as far as we know, the exact mechanisms remain unknown. It is moreover admitted that TMD may influence the cervical spine function inducing impairment in range of motion and potentially worsening CD [119].

Post-traumatic stomatognathic system and associated symptoms

TMD may result from various stomatognathic conditions including CCT [107]. Post-traumatic TMD is moreover the most investigated type of stomatognathic system impairment. The diagnosis relies mainly on a history of temporomandibular joint complaints associated with movement, local discomfort, and a contributive physical examination [107].

According to a recent systematic review, dizziness and vertigo affect approximately 40% and 22% of adult patients with TMD [118]. Viziano and colleagues [120] notably reported in an observational study that patients affected by TMD and CD tend to show worse postural performances than subjects affected by CD or TMD alone. Currently, there is no clear evidence concerning the effect of TMD therapy on dizziness and vertigo [121]. Further studies are therefore needed to assess whether TMD may result independently in vertigo or dizziness to target proper specific interventions.

Migraine, tension-type headaches, and neck pain are also commonly associated with TMD [122]. Headaches and neck discomfort have been reported to be the most common presenting symptoms along with TMD after WI [51]. Some authors showed that these symptoms are significantly worse when TMD is due to WI and are associated with higher neck pain intensity [123]. Based on 2 case-control studies done on TMD patients, Lee and colleagues [108,109] reported a significant increase in neck pain and headache in post-WI

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Table 3. Main clinical associated conditions to temporomandibular disorders

Clinical conditions
- Otological symptoms: ear fullness, earache, otalgia, tinnitus, hearing loss [118]
- Vestibular-like symptoms: dizziness and vertigo [118]
- Head and neck disorders: migraine, tension-type headache, neck pain or limitation in range of motion [122-124]

TMD patients compared to patients with TMD from idiopathic causes or other sorts of trauma TMD associated clinical conditions are summed up in **Table 3** [118,122-124].

Treatment for TMD, migraine, tension-type headaches, and neck pain, particularly when post-traumatic, are multimodal, including manual therapy, exercises, patient pain education and counseling [24,100,102,124-126]. However, to date, there is very low certainty that there is a beneficial effect of physical therapy for TMD on concomitant headache intensity compared to control interventions [126]. Treatment of TMD and its related headaches have furthermore been shown to have poorer outcomes when associated with WAD [34]. We assume that headaches, neck pain, and TMD after CCT need to be considered from a global perspective and as plausible intercorrelated entities.

CONCLUSION

mTBI and WI may lead to various symptoms and long-term disabling conditions namely PCS and WAD. Many recommendations on how to treat their consequences have been proposed in the literature so far. However, a lot of ground remains to be covered in the understanding of the real consequences of these 2 very similar conditions.

Ocular sensorimotor, vestibular, cervical proprioceptive, and stomatognathic systems, all involved in postural control, may be damaged by CCT. Impairment of these systems has been shown to lead to various conditions such as headache, dizziness, visual disorders, or neck pain, all of which are also reported by PCS and WAD patients. Considering the post-traumatic impairments of the structure involved in postural control in the treatment of PCS and WAD-related symptoms remains at best promising but lacks evidence. Yet, few or no studies have considered the evaluation and/or treatment of these widely interconnected systems from a gathering perspective. We proposed that future research needs to consider all these systems after CCT from a postural control perspective.

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